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FATALITY ASSOCIATED WITH BENZEDRINE INGESTION:

A Case Report

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Since benzedrine[1] (amphetamine) was introduced as a therapeutic agent by Prinzmetal and Bloomberg (1) in December 1935, it has been widely and extensively used in the treatment of narcolepsy, hypotensive states, obesity and various depression states. Fatalities associated with the ingestion of benzedrine are of such infrequent occurrence as to warrant reporting. The literature to date contains reports of but three such instances.

Smith, (2) in 1939 reported the death of a 25-year-old college student who regularly took 5 mg. before examinations and who had taken probably 30 mg. during the few days prior to his sudden collapse and death while taking a written examination. An autopsy performed four and one-half hours after death provided no anatomical explanation other than dilatation of the right auricle, and gastric and splanchnic dilatation. Chemical analysis of the gastric contents (by a method not disclosed in Smith's report) demonstrated the presence of about 0.25 mg. amphetamine (benzedrine) sulfate.

Sudden collapse with coma and death in a 1-year-old child following the accidental ingestion of a minimum of 40 mg. benzedrine sulfate and an unknown number of ferrous sulfate tablets was reported by Hertzog *et al* (3) in 1934. The anatomic diagnosis was recent hemorrhage in the gastric wall and adrenals, and edema of the lungs (examination of the brain was not permitted). Death was attributed to benzedrine sulfate poisoning.

Gericke, (4) in 1945, reported a case of death in a 36-year-old soldier following the ingestion of an estimated 120 mg. benzedrine sulfate taken with suicidal intent. The patient had a long history of psychoneurosis and alcoholism. The major findings at autopsy were subdural and subarachnoid hemorrhage, petechial hemorrhages of the cerebrum, hemorrhage of the pons, congestion of the lungs, spleen and kidneys, and pulmonary edema. The immediate cause of death was attributed to the subdural and subarachnoid hemorrhages.

Case Report

J. H. (Hospital #156938) a 35-year-old white male was admitted to the Delaware Hospital, on September 21, 1948, at 4:50 P. M. with the complaints of abdominal pain, malaise and both auditory and visual hallucinations. The history as ascertained from hospital and police records is as follows:

The patient, a known vagrant with a record of two previous arrests for drunkenness, was apprehended on the street shortly after noon on September 21, 1948. He was taken to another hospital in the city for examination preliminary to being jailed on a charge of vagrancy. During the examination, he complained of seeing "large animals" on closing his eyes and of hearing "radios" in his ears. He volunteered the information that he had ingested part of the contents of a "benzedrine inhaler" (SKF), containing 250 mg. racemic amphetamine, on the advice of a friend who told him that it would help the symptoms of "hangover" and give him a "kick". The patient appeared jittery and had a blood pressure of 145/80. Since he did not appear ill otherwise, he was released from the hospital and taken to police headquarters. There, he continued to complain of abdominal pain

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1 Trade-marked name of racemic amphetamine, S.K.F.

and hence was brought to the Delaware Hospital by the police for re-examination. At this hospital, the patient stated that he had ingested the contents of *two* "benzedrine inhalers" and he admitted having drunk about 2 pints of wine and 8 "beers" in the previous 24 hours.

Physical examination disclosed a heavy set white male who did not appear seriously ill but was very apprehensive and hyperirritable. The pulse was 120/min.; temp. 99.8°; and respirations 32/min. Positive physical findings included flushing of the face, recently sutured small lacerations of the face and forehead, a subconjunctival hemorrhage on the left side and a firm, nontender liver edge palpable 2-3 fingerbreadths below the costal margin. All reflexes were hyperactive and equal, and there was a fine tremor of the hands and tongue. The pupils were small, round and reacted equally to light stimulation. The lungs, heart and abdomen, except for the liver, were negative. Laboratory studies revealed a hemoglobin of 11.4 gms. and a WBC of 3,300 (53% segmented polys, 29% non-segmented polys, and 18% lymphocytes). The serology (Mazzini) was negative.

The patient was placed on a regimen of bed rest, forced fluids and sedation with sodium amytal and chloral hydrate. He complained of intense thirst and drank large quantities of fluids but vomited frequently. He appeared to be improving slowly till the morning of September 23, 1948, when his condition appeared to have changed suddenly. Icterus and an ashen cyanosis were noted. He became irrational and developed signs of circulatory collapse. Oxygen therapy, epinephrine and intravenous fluids were started and blood had been ordered when the patient expired unexpectedly. Just prior to death the temperature had risen to 104° (R). An autopsy was performed 1 hour and 20 min. after death.

REPORT OF AUTOPSY

The body is that of a muscular white male appearing the stated age of 35 years. The external examination is not remarkable except for moderate icterus of the skin and sclerae, a subconjunctival hemorrhage and small sutured lacerations of the face and forehead.

Internal examination shows ecchymoses and petachiae scattered throughout the mesentery, omentum, mediastinum and over the serosa of the bowel and pericardial surfaces. The liver edge extends 9 cm. below the xiphoid and 5 cm. below the costal margin in the right midclavicular line. The stomach and intestines are distended with gas and fluid. The *heart* weighs 425 grams and there are subepicardial petachiae. Microscopically there is slight myocardial edema and congestion. The *lungs* show both gross and microscopic evidence of edema and congestion. The *spleen* weighs 350 grams and is firm with smooth surface and rounded edges. There is obscuring of the structural markings. Microscopically there is marked congestion. The *kidneys* show a peculiar tan discoloration grossly and cloudy swelling microscopically. There is edema of the submucosa of the *small bowel.* The *brain* weighs 1475 grams and shows gross evidence of slight edema and capillary congestion which are also evident on microscopic examination. There are no hemorrhages. The *liver* weighs 2600 grams and has a smooth, glistening capsule with an unusual reddish orange color. The edges are rounded and the consistency uniformly rubbery. The cut surface bulges slightly and has a greasy appearance and bright reddish orange color with dark red specks. On exposure, the color changes to a striking brick red.

Microscopic examination of the liver (Fig. 1 and 2) reveals lobular disarray. The parenchymal cells show marked fatty change with the central and mid-zones showing greater change than the peripheral zone. In the central regions of the lobules the cell outlines are indistinct and the cytoplasm is replaced by fat vacuoles. The remaining nuclei are pyknotic. The microscopic appearance of the liver differs from that of the ordinary type of severe fatty metamorphosis in that central necoris is added. The cells in the peripheral zone are intact but also show less extensive fatty change with pyknosis and karyorrhexis of the nuclei. "Signet-ring" cells are abundant. There is a peri-vascular infiltration of both chronic inflammatory cells and polymorphonuclear leukocytes. The vessel walls appear partially necrotic and are actually infiltrated with the inflammatory cells. There is a suggestion of prominence of the bile

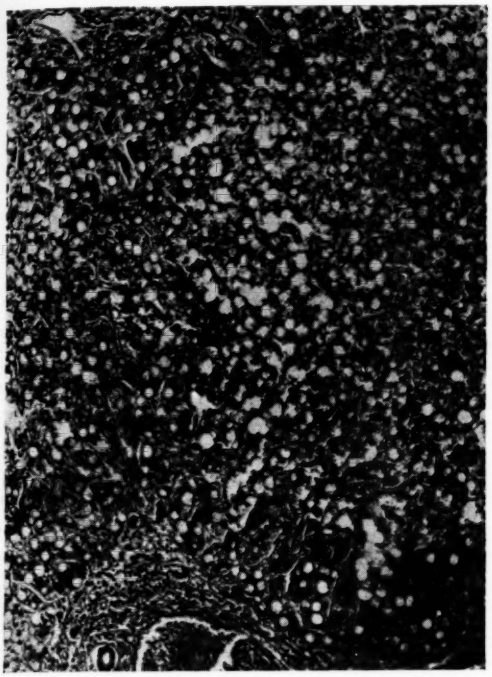

Figure 1. Section of liver showing marked fatty change and necrosis of hepatic cells (x115).

canaliculi in the better preserved liver cords. There is no evidence of perilobular fibrosis, hepatic cell regeneration or bile duct proliferation.

The liver, heart and brain were tested for the presence of benzedrine (b-phenylisopropylamine) using the color reaction of Beyer (5). Positive tests were obtained in each case, whereas negative tests were obtained for cirrhotic liver tissue from another source. This test is based upon the extraction with n-butanol of the red color formed by the action of sodium hydroxide on the Benzedrine—p-nitrobenzenediazonium chloride interaction product. Related compounds (primary amines) also give this same color reaction.

PROCEDURE*

Ten grams of the tissue are macerated with 50 ml. of 0.5 N hydrochloric acid. This suspension is shaked vigorously to extract the amine and filtered. The filtrate is rendered alkaline by the addition of 10 ml. of 20% sodium hydroxide, and extracted with 25 ml. of ether. The amine is again extracted from the ether phase with 5 ml. of 0.5 N hydrochloric acid and the acid extract evaporated to dryness on a small watch glass over a steam bath. The residue is dissolved in 1 ml. of water and transferred to a 50 ml. volumetric flask.

A solution of p-nitrobenzenediazonium chloride is prepared as follows: p-Nitroaniline (0.69 gm.) is triturated with 3 ml. of concentrated hydrochloric acid. This suspension is then diluted to 100 ml. of water. To 5 ml. of this solution is added 1 ml. of concentrated hydrochloric acid and the resulting solution is chilled in an ice bath. Three milliliters of 0.7% aqueous sodium nitrite is added. After standing in the ice bath for 6 minutes the solution is diluted to 100 ml. with ice cold water.

Five milliliters of the above solution of p-nitrobenzenediazonium chloride is added to the solution of the residue in the 50 ml. volumetric flask. Five milliliters of 1.1% aqeous sodium carbonate is then added drop by drop with continuous mixing. After standing for 15 min., 1 ml. of 10% sodium hydroxide is added drop by drop with continuous mixing.

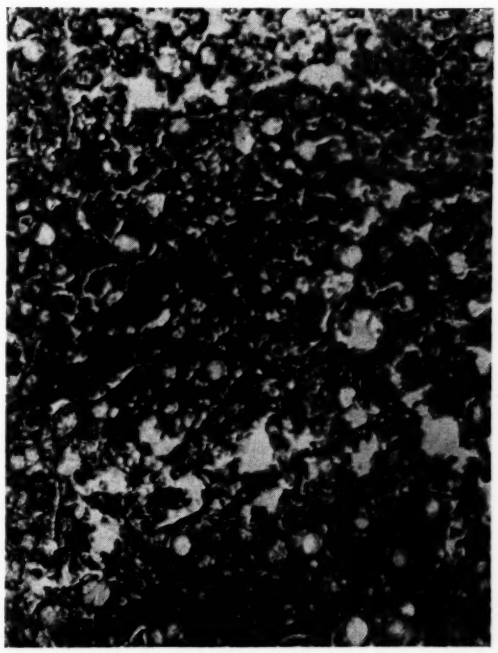

Figure 2. Higher power (x264) of same area.

* The authors are indebted to Miss Martha Withers, MT, for her assistance in the performance of the chemical procedures, and to Dr. Herbert W. Taylor of Smith, Kline & French Laboratories for technical advice.

After standing for 10 min. this solution is extracted with 10 ml. of n-butanol.

The presence of benzedrine or a closely related compound is revealed by the formation of a red color upon the addition of the sodium hydroxide which is extracted with n-butanol. In the tests performed with the three different tissues from the patient, color intensities comparable to those obtained from 1 ml. of a 0.003% benzedrine sulfate solution were obtained whereas the end product with the control liver tissue was colorless.

DISCUSSION

According to the manufacturers (Smith, Kline & French Laboratories) "benzedrine inhalers", when new, contain 250 mg. racemic amphetamine plus menthol. It is estimated that this patient ingested a maximum of 500 mg. of benzedrine although probably less. Admittedly the patient's history is entirely unreliable as evidenced by his two different accounts as to the amount of drug ingested. Yet the chemical demonstration of benzedrine in the body tissues more than 48 hours after its ingestion indicates that a comparatively large quantity of the drug must have been consumed; or else, because of a previously damaged liver, the patient was unable to "detoxify" the drug at the normal rate. Regardless, the estimated maximum dosage would be about 6 mg./kilo body weight.

Ehrich, Lewy and Krumbhaar (6), from their experimental studies on the toxicology of benezdrine sulfate in animals, found the minimum lethal dose in their most susceptible animals to be about 5 mg./kilo and deduced that the MLD in man is probably not below that level. They cite the case of a 67 year old man who took between 300 and 800 mg. of benzedrine (probably 450 mg.), and another, a manic-depressive, who took 350 mg. on one occasion and later, according to the patient, ingested *20 gm.* over a two day period. Both men recovered uneventfully. Waud (7) described a series of remarkable experiments on a young man weighing 97 kilograms who inhaled 650 mg. in a 4-6 hour period of continuous inhalation without serious effects. Waud estimated that his subject actually absorbed from 400 to 500 mg. Robinson (8) reported the case of a man taking as much as 250 mg. benzedrine sulfate daily for an undisclosed length of time and that of another man who took 160 mg. daily for 3 weeks with neither of them showing ill effects.

That benzedrine sulfate may be administered in large doses over prolonged periods of time without demonstrable evidence of toxicity was shown by Bloomberg (9) in his follow-up studies of three narcoleptic patients, two of whom took 70 mg. daily for 2 years 8 months and the other, the same amount for 1 year 8 months. Very thorough physical and laboratory studies failed to show evidence of toxicity. Impressive doses have been used in the treatment of obesity and psychiatric problems in children (10).

Critical analysis of the three fatal cases reviewed and the one herein reported leads to the impression that the role of benzedrine sulfate as the sole causative factor must be held questionable. The ingestion of benzedrine in all four of these cases is accompanied by modifying extraneous factors. In the case of the college student reported by Smith, there was, besides the ingestion of an estimated 30 mg. per day for a few days, the presence of "examination nervous tension" plus fatigue plus the effects of a large meal. The child reported by Hertzog had ingested, besides benzedrine, an unknown large number of ferrous sulfate tablets. Ferrous sulfate is not an entirely innocuous substance (11). Finally, the ingestion of benzedrine in Gericke's case, as in our own case, was associated with a known long history of alcoholic overindulgence.

The lack of uniformity in the pathologic findings at autopsy throws still further doubt as to the role played by benzedrine in these fatalities. The pharmacology of benzedrine sulfate has been thoroughly reviewed by Ivy and Krasno (12), but our knowledge of the pathologic changes produced by benzedrine toxicity is derived solely from studies on animals. Ehrich *et al* (6) found that animals dying from benzedrine overdosage showed marked dilatation of the heart; congestion of the liver and kidneys; either congestion or contraction of the spleen; air in the stomach and intestines; and, in some animals, subpleural and pericardial hemorrhages as well as marked and sharply delimited constrictions of the small intestines. The only histological

lesions they found in the thoracic or abdominal organs that could be connected with the action of the drug were necrosis in the liver and spleen. Most of these animals had received extra large doses. The animals' brains showed the characteristic signs of acute intoxication: namely, venous stasis, perivenous hemorrhages in the meninges, white matter of the cerebral hemispheres and cerebellum, and toxic degeneration of the nerve cells in these regions. Smith's case revealed only functional changes. The single common denominator in the remaining cases appears to be a hemorrhagic diathesis although the hemorrhages occurred in quite different localities. There is little doubt that the liver in our case shows the pathologic changes typical of an alcoholic fatty liver. We believe, however, that the presence of central necrosis is due to the added toxic effect of benzedrine sulfate. This finding is in conformity with the liver necrosis occasionally seen in animals receiving extra large lethal doses of this drug. Our conclusion is that benzedrine was merely a contributory factor in the patient's death and in the presence of a normal liver would probably not have resulted in a fatal termination.

SUMMARY

1. Fatality associated with the ingestion of benzedrine sulfate has previously been reported but three times. These cases are reviewed and an additional case is reported.

2. A procedure for the qualitative detection of benzedrine and related compounds in tissue is described.

3. The importance of benzedrine sulfate as a causative factor in the fatal cases attributed to its ingestion is discussed.

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ROENTGEN THERAPY OF SUBDELTOID BURSITIS

REVIEW OF 235 CASES

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This is a report on 235 cases of subdeltoid bursitis (sub-acromial bursitis, peritendinitis calcarea, pararthritis of the shoulder joint, etc.,) treated by roentgen therapy during the past five years at the Delaware Hospital. Questionnaires were sent to 442 patients requesting the following information: duration of symptoms, previous attacks, other forms of therapy employed, response to roentgen therapy—whether complete relief of pain, marked improvement, slight improvement, or no response at all, time elapse before relief of pain, recurrence of pain, and whether or not radiographs were taken of the involved shoulder.

It was not deemed necessary to rely on radiographs of the shoulder taken before treatment. The diagnosis in the majority of the cases was made entirely on clinical findings. The most common signs and symptoms in the acute cases (either first attacks, or acute exacerbation of a chronic condition) were pain referred to the insertion of the deltoid muscle, radiating into the neck, down the arm or to the hand, occurring at rest and either on voluntary or passive motion, and being more severe at night. Point tenderness was most commonly noted below the acromion process, just over the greater tuberosity of the humerus. Swelling may or may not be present and redness is absent. The sub-acute to chronic forms may exhibit some point tenderness, but mainly gradations of restriction of motion, particularly in abduction or rotation of the humerus at the shoulder joint. Barton Young reported approximately the

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same incidence of calcification in his grouping of acute, sub-acute, and chronic cases which was 43. plus %[1]. This was surprising because originally, we believe, it was thought that the acute cases may not exhibit chiefly radiographic changes, and these represent those cases in which the calcification may be microscopic or collection of tendon debris showing no opaque shadow by x-ray[2].

The cases reported in this series were divided into acute, sub-acute, or chronic groups according to duration of symptoms, i.e. respectively up to one week, up to two months, and over two months. The age grouping is demonstrated in Table I.

TABLE I

AGE GROUPS OF PATIENTS TREATED FOR SUBDELTOID BURSITIS

Years	20-29	30-39	40-49	50-59	60-69	70+
Acute	3	20	18	12	3	2
Subacute	5	17	16	20	6	2
Chronic	3	13	25	12	8	0

Two techniques of treatment were used: low voltage and deep. The low voltage treatment consisted of a total dosage of 400R in air in daily doses of 100R, 150R, and 150R. The physical factors were: 120 KVP, 10MA, and 1.5 mm. al added giving equivalent of 4mm AL filter at 30 cm. target skin distance. One portal was used covering a circular area of 10 cm. in diameter aiming down, in and back, just under the acromion process. The deep therapy consisted of a total dosage of 600R in air, 200R on alternate days for three doses, using 200 KVP, 15 MA, 0.5 cu., 0.5 al, at 50 cm. target skin distance covering a circular area of 12 cm. in diameter and aimed in the same manner as the low voltage treatment.

Although a great many patients were relieved of symptoms after the second or even the first treatment, the series of three were completed to better evaluate the duration of relief of pain or recurrence, and to make the effect more lasting.

We shall divide the results primarily into two groups: those receiving a total dose of 400R (in air) and those receiving a total of 600 (in air). In the 400R group, there were a total of 183 patients treated. Forty-five of these were classified as acute, with symptoms up to one week; 61 subacute, with symptoms up to two months; 77 chronic with symptoms lasting over two months. The results in the

TABLE II

183 PATIENTS TREATED WITH LOW VOLTAGE THERAPY—400R (IN AIR)

	Acute (Symps. up to 1 wk.) Cases	%	Subacute (Symps. up to 2 mo.) Cases	%	Chronic (Symps. over 2 mo.) Cases	%
Complete Relief	33	73.3	38	62.3	31	40.2
Marked Improvement	8	17.6	12	19.7	12	15.6
		90.9		82.0		55.8
Slight Improvement	2		5		12	
No Response	2		6		22	
	45		61		77	

400R group are totaled in Table II. In the acute cases 33 or 73.3% had complete relief of pain; 90.9% (33 complete relief, 8 marked improvement) were the results of grouping those who had complete relief and those who noticed marked improvement. The subacute cases responded 62.3% and 82% using the same classifications, and the chronic cases 40.2% and 55.8%.

In the 600R group, a total of 52 patients were treated, four acute, ten subacute, and 38 chronic, the results of which are evident in Table III. We considered the small number

TABLE III

52 PATIENTS TREATED WITH DEEP THERAPY—600R (IN AIR)

	Acute (Symps. up to 1 wk.) Cases	%	Subacute (Symps. up to 2 mo.) Cases	%	Chronic (Symps. over 2 mo.) Cases	%
Complete Relief	4		3	30	9	23.7
Marked Improvement	0		3	30	9	23.7
				60%		47.4%
Slight Improvement	0		3		9	
No Response	0		1		11	
	4		10		38	

of cases in the acute and subacute divisions to be inconclusive, but there is a sufficient number of chronic cases to compare those treated with low voltage therapy. Of the 77 chronic cases receiving low voltage therapy, 31 patients or 40.2% welcomed complete relief and these plus the twelve patients showing marked improvement give a percentage of 55.8%. Comparing this with the total of 38 chronic patients who received deep therapy, 9 or 23.7% had complete relief and together

with the 9 who showed marked improvement, this gives a total of 47.4%. This, we think, gives some evidence that deep therapy may not be of any more value in the chronic cases than is the low voltage method of therapy.

The patients who claimed complete relief from both types of therapy were 118 in number. The relief was noted to occur in the majority of the cases almost immediately—some after first or second treatment—others two to three days after the series of treatments were completed. Of the 118 patients who were placed under the heading of complete relief, all but 16 experienced this relief in a period of up to one week. These 16 people, however, were relieved not later than two to three weeks.

The patients were instructed not to use heat or rubbing liniments while receiving treatment. Until pain disappeared, they were advised to use ice packs only if thought necessary. No active or passive motion of the involved shoulder was advocated until pain had subsided, and then graduated exercises were advised. Quite a few patients experienced increase in symptoms after the first treatment before relief was noticed, and therefore, were forewarned that this might occur.

CONCLUSION

As observed in other papers written on this subject, the best results from roentgen therapy were obtained in the acute cases of subdeltoid bursitis, either first attacks or acute exacerbation of the chronic condition. The sooner therapy was instituted, the quicker the response, and the better the chance of complete relief of symptoms were noted.

Good comparable results were obtained using a lower voltage technique with only one portal, and smaller daily dosage of R units with lower total dosage of R units than have been reported previously.

From the results obtained on this series of cases, it would appear that low voltage roentgen therapy for subdeltoid bursitis is just as effective as deep therapy, whether acute, subacute, or chronic.

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METABOLIC OUT-PATIENT CLINIC SURVEY

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The Metabolic Clinic of the Delaware Hospital was established in 1930 for the ambulatory care of patients with diabetes mellitus and other metabolic disturbances. The present report is a statistical study of the records available for 1947, 1948, and the first four months of 1949. The records of many patients are not complete, and the number of patients included in the study is not large. For these reasons no definite statistical conclusions can be drawn. It is felt, to be of interest, none the less, to show the general trend of our findings, and perhaps to point the way toward improved statistics from this clinic in the future.

To overcome the difficulty of securing incomplete records, a guide was designed for the physician's use in the Clinic. This is a flow chart, listing the following examinations to be done: *quarterly*—a complete urinalysis, capillary fragility, fundoscopic examination, examination of the vascular tree. *semi-annually*—blood urea and cholesterol determinations, diet check with the hospital dietician. *annually*—chest x-ray, complete physical examination and resume of the patient's progress for the year. In our Clinic, which is attended by different groups of house officers, this chart aids in coordinating the care of the diabetic patient.

During the period covered by this report (Jan. 1947 to May 1949) a total of 139 patients have been seen, for a total number of 1556 visits. Of this number, 85 have been cases of diabetes mellitus; 54 have had some other metabolic or endocrine disturbance. During 1947 the average number of patients seen at the weekly clinic was 10.6. The number increased in 1948, and during 1949 the average number has been 18.

TABLE I

Under 11 years	1
11-20	3
21-30	3
31-40	8

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41-50	15
51-60	29
61-70	15
71-80	8
Over 80	0

Table I shows the age distribution of the diabetic patients. The greatest number fall in the decade from 51 to 60 years, but it is noteworthy that there are seven patients under the age of 30. Curiously there have been 64 female patients, and only 11 male patients. It seems likely that this disparity is influenced more by sociologic and economic factors than by the true incidence of diabetes.

TABLE II

Under 10 years	2
11-20	3
21-30	4
31-40	14
41-50	18
51-60	23
61-70	8
71-80	1
Over 80	0

Table II shows the distribution of the age of onset of clinical diabetes in our patients. Here, as would be expected, we see an increasing incidence in the younger age groups. It is hardly necessary to point out, as Joslin[1] has so often done, that diabetes should no longer be thought of as a disease of old age.

TABLE III

1900-1925	2
1926-1930	4
1931-1935	5
1936-1940	12
1941-1945	20
1946-1949	29

Some ideas of the duration of diabetes in our patients can be derived from Table III which shows the date of onset of diabetes. One patient's onset occurred before the discovery of insulin, one patient's dates from 1923. Twenty-three patients have had their illness for more than nine years.

TABLE IV

Under 100 lbs.	3
101-125	15
126-150	20
151-175	18
176-200	11
201-225	5
226-250	4
Above 250	1

Much has been written about the correlation of diabetes mellitus[1] with obesity. Table IV lists the current weights of our diabetic patients, without correlation as to height. It is probably significant that in a group predominately female, over 10% weigh more than 200 pounds, and about half weigh more than 150 pounds. We have not attempted to correlate severity of diabetes with obesity, but it may be of interest to mention the three patients who weigh less than 100 pounds. One is a child of 9 who requires 56 units of mixed insulin daily; the other two are adults, 48 and 51 years old, who require 50 and 40 units of insulin daily, respectively, for adequate maintenance.

TABLE V

Retinopathy	12
Cataracts	6
Corneal scarring	2
Congenital lens defect	1
Retinitis proliferans	1
Optic atrophy	1
	22

Table V lists eye findings in our diabetic patients. Unfortunately 63 patients have not had adequate eye examinations. 22 patients were found to have various degrees of ocular disability, the largest number, 12, having some stage of the vascular retinopathy associated with either hypertension or arteriosclerosis, and diabetes. No correlation has yet been attempted between retinopathy and other factors such as control of diabetes.

27 of our patients have consistent elevation of blood pressure, the systolic being above 140 and the diastolic above 90 mm. Hg. This represents a rather high incidence of hypertension for such a group.

There are 16 patients in this clinic who have definite symptomatic peripheral vascular disease, six who probably have peripheral vascular disease as evidenced by moderate symptoms but in whom detailed studies such as oscillometric readings and soft tissue x-rays have not been done. Of these 22 patients, six have hypertension, four have retinopathy. One patient has had bilateral mid-thigh amputations for peripheral vascular disease, one has had a unilateral mid-thigh amputation.

The records show 10 patients who have de-

finite coronary artery disease as manifested by electrocardiographic evidence of previous myocardial infarction, or by symptoms of angina with confirmatory electrocardiographic evidence. Five other patients are listed as probably having coronary artery disease.

Hospital records for the year 1947 and the first eight months of 1948 showed 13 diabetic patients with acute or old myocardial infarction. Some of these patients had been followed or subsequently have been followed in the Metabolic Clinic. Of these 13 patients nine were male and four female. This is a ratio of males to females of 2.25:1, considerably lower than the reported ratio in non-diabetic patients. This low ratio is in keeping with other reported series of diabetic patients with myocardial infarction. The average age of the male patients was 61 years, that of the females 57.7 years. Among the patients with myocardial infarction, only three required more than 30 units of insulin daily, and none of these required more than 35 units daily for satisfactory control. Indeed, three of them were controlled by diet alone. Thus, none of the patients with myocardial infarction had severe diabetes. This has generally been true of our patients with peripheral vascular disease. These findings are in accord with the larger published series which have been unable to relate the manifestations of arteriosclerosis with severity of diabetes.

The findings set forth above do not purport to be adequate for statistical analysis, for the number is small, and in many cases the record has been incomplete. We have, however, been interested to find that in general the trend of our statistics is in the direction taken by other larger and more complete reports, namely: that as coma becomes less and less prominent as a cause of death in diabetics, and consequently the duration of life with diabetes lengthens, arteriosclerotic complications and their prevention form the main problem to be dealt with by clinicians; that these complications are ocular, peripheral vascular, and cardiovascular; that at least the peripheral vascular and cardiovascular complications are not particularly associated with severe diabetes.

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CLINICOPATHOLOGIC CASE RECORD

JOSEPH A. ELLIOTT, M. D.,*
M. MARGARET SMITH, M. D.,*
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PRESENTATION OF CASE**

DR. SMITH: A 48 year -old Negro male was admitted to the hospital on 9-12-47 with the chief complaints of: trouble getting his breath, abdominal swelling, pain in right chest—posteriorly, and ankle edema of two months duration.

History dates back to approximately 7 years ago at which time patient was hospitalized for an episode of dyspnea, ascites and ankle edema. In the succeeding years exertional dyspnea became gradually more marked. For over 2 years patient had been receiving diuretics.

Systemic review reveals weight loss of 15 pounds in past month, intermittent fever of 1 month duration. No chills, night sweats or cough. System otherwise negative.

Past medical history reveals exertional tachycardia since childhood. No rheumatic fever, pneumonia or other illnesses.

Physical examination reveals a temperature 101°F, respirations 28, pulse 110, blood pressure 110/80 right arm and 110/50 left arm. Patient is an acutely ill middle-aged dyspneic colored male. There was distention of the veins of the neck, arms, and lateral thorax, a small left supra-clavicular node. Chest expansion limited on the right. Left lung normal to percussion and auscultation. There are diminished breath sounds throughout the right chest, absent posteriorly. Tactile fremitus, absent over right chest. Percussion note dull to flat over that area. Heart reveals point of maximum impulse at 6th interspace, left, just lateral to nipple line. Rhythm regular. Rate 110. No definite murmurs. Pericardial friction rub heart at the apex. Abdomen is distended. Liver palpable (non-tender) about 3 to 4 fingers below costal margin. Spleen, palpable 2 fingers below costal margin. Peristalsis normal. Reflexes absent in lower extremities.

Examination of blood showed a red blood count 4.1, hemoglobin 12 gms. white count 2,800 to 5,800, polys seg. 67%, polys non-seg.

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** Delaware Hospital Case No. 148835, presented at Staff Clinical-Pathological Conference.

19%, lymphocytes 8%, Monocytes 6%. Urine showed occasional albumin and clumps of pus cells. Culture showed Mycin sensitive hemolytic E. coli and atypical coliform bacilli. Wassermann and Kahn negative. Urea nitrogen 10.6 mgm%, albumin 2.6 gms. globulin 3.5 gms., cephalin cholesterol floc. plus 1, prothrombin 78%, Congo red no dye absorbed in 1 hour. Stools negative for occult blood. Febrile agglutinins typhoid 0 1:320, typhoid H 1:80, brucella abortus 1:40 and proteus OX 19 1:20. Chest fluid clear on first tap, subsequent taps blood tinged; cultures on each occasion showed no growth and were negative for tumor cells. Blood cultures negative. Sputum and gastric washings negative for acid-fast. Venus pressure 9-23-47, 160 mm H_2O, and 10-1-47, 230 mm. H_2O. Circulation time, arm to tongue 28.5 sec., arm to lung 19.6 sec., lung to tongue 8.9 seconds.

Electrocardiogram shows persistent low voltage. Bigeminal rhythm. Sinus tachycardia on various tracings. X-rays show right pleural effusion, degree dependent upon thoracentesis.

Course in hospital. Repeated thoracenteses done. On 10-10-47 a biopsy of inferior cervical node was done under local anesthesia. On 10-11-47 a peritoneoscopy was done. Straw colored clear fluid was aspirated (1 pint). No specific lesion was seen due to numerous adhesions. Subsequent course was downhill; loss of appetite; apparently increasing jaundice terminally; marked dyspnea. On the 43rd hospital day, after patient vomited a small amount of yellow frothy blood flecked material, respirations became extremely shallow, rapid, and patient expired. An autopsy was performed.

DIFFERENTIAL DIAGNOSIS

DR. ELLIOTT: I think probably we must start with an original premise here, and say that among the 30 findings of variable significance, those that are most markedly important stand out quite well, and others, of more vague significance, must be explained by more difficult means.

Initially, I think it would be well to comment on three pertinent findings as evidenced by the electrocardiogram and physical examination. That of the EKG is a persistent low voltage. Those of the physical examination are ascites and an increased venous pressure. This triad is almost pathognomonic of chronic constrictive pericarditis. I think that fact should be put down initially.

Now, chronic constrictive pericarditis usually begins as an acute pericarditis, commonly of an unknown origin. Those organisms that are most frequently involved are those of the pyogenic group and M. tuberculosis. In my reading I found, though I am not sure that this is absolutely correct, that the usual age of occurrence of constrictive pericarditis is at 45 years. This patient was 48 years old. The relative incidence is 3 to 1 for males, and I might add it is uncommon as a clinical entity, which I assume predisposes some underlying disease as the cause. In the light of other findings here listed it is worth stating that in the final stages of chronic constrictive pericarditis there is eventual obstruction of the hepatic veins with consequent enlargement of the liver. Congestion, and cirrhosis, occur too. One also sees perihepatitis and perisplenitis. These are terminal findings and are described as part of the findings of Pick's disease, the "sugar-coating" appearance, so called.

We must consider the findings of weight loss, intermittent fever, and splenomegaly of significance. This man is 48 years of age. He has had a persistent weight loss prior to his hospital admission. One probably could say with the reasons that follow in the course of this discussion that this man could have had and probably did have an old tuberculous lesion. (1) He entered the hospital with acute dyspnea (which could have occurred as a part of his constrictive pericarditis). (2) He had one-side pleural effusion which could probably be best explained by the fact that he has an active pneumonic process conceivably of tuberculous origin. (3) He was here some 43 days and apparently became progressively worse. (4) His thoracentesis showed straw colored fluid which may have been an exudate, though this is not known. (the usual appearance of fluid from a thoracentesis in a patient with tuberculous pneumonia or some other tuberculous process is turbid. (5) Organisms are commonly not returned and seen by stain and microscopic examination in a person with tuberculous process. It is com-

mon and routine probably, in a large number of places, that this fluid, if suspected to be of tubercular origin, be injected into guinea pigs. This is not reported as having been done. It would be nice if we did know that and the results.

Then there is another finding which warrants some explanation. We have in the protocol two results of febrile agglutinins, and without some study I was at quite a loss to account for these. It is significant to note that there is a rise in titre in all the reported febrile agglutinins while he had been in the hospital. It is also significant to note that the typhoid 0 agglutinin as reported is above a significant titre. I think 1 to 300 is probably the limit below which one does not attribute much significance. This is 1 to 320. We do not know whether this man has ever had typhoid fever, nor do we know whether he has ever been immunized against typhoid. I have one explanation that I feel may answer this problem. I originally stated that this man probably had an active tuberculous process, maybe a tuberculous pneumonia with a pleural effusion associated on the right, as he had right sided chest pain as noted also. On his third day after admission it is noted that there is an elevation in his febrile agglutinins which was not present on his admission date (negative). There is no comment whether his condition became worse at that time. It is known that in miliary tuberculosis there is a significant alteration in the febrile agglutination picture. This author from which I found this information does not describe what the alteration is, he said that it is significant, and that it most comonly involves the typhoid and the para-typhoid agglutination groups.

There is no comment made as to brucella or proteus OX19 as listed here.

Miliary tuberculosis is commonly described under three types, namely, typhoid, pulmonary and meningeal. The meningeal form is usually heralded by the presence of fever, anorexia, vomiting, headaches, prostration, and commonly, nose bleeds. In addition there is lethargy occasionally with persistent meningeal irritation, photophobia, occasional delirium, stiff neck with positive Kernig's and Brudzinski's signs, nystagmus, convulsions, strabismus, etc. The spinal fluid is significant in the fact that often the organism can be returned in the fibrin clot of the spinal fluid itself after it has been allowed to stand. There is also a characteristic increase in lymphocytes and a decrease in chlorides. I do not think that this form exists here. In the pulmonary type there is a relative disproportion between the rapidity of respiration and the height of the temperature. This man's temperature, I am told, was never above 102. His respiratory rate is 26 on admission. Whether that is a relative disproportion is difficult to say. There is severe dyspnea and occasionally cyanosis with this form, and there is demonstrable evidence by X-ray. The tuberculosis organism is frequently recovered in the gastric washings, sputum, and occasionally, the feces. Death is common in 1 to 3 months with this type of miliary tuberculosis. The most interesting in this group I saved until last, The typhoidal form is commonly heralded, as in the meningeal type, with fever, anorexia, headache, and prostration. As these symptoms progress there is dyspnea and commonly splenomegaly. That is also listed here as being a physical finding. Blood cultures are commonly sterile. The stool and urine examinations, frequently are reported showing the usual organisms. And as I said before, and probably most significant in this case, agglutination reactions of typhoid and para-typhoid are normal or misleading.

Then we are faced with the fact that a biopsy of a lymph node was done and we know also that this patient was jaundiced terminally. Could this be a generalized lymphadenopathy secondary to a miliary tuberculosis? We must answer yes.

He had a peritoneoscopy done and a straw colored fluid was found, but of a very small amount, possibly a pint. Commonly in tuberculous peritonitis, which I think also must be considered, one sees the presence of many abdominal adhesions between loops of bowel and a variable amount of free fluid in the abdomen. At its onset there is more, but generally as this disease progresses less fluid is able to be returned, probably because it becomes loculated. I think probably we should also consider the fact that he had tuberculosis peritonitis. However, we must consider the following as against a pure, uncomplicated

tuberculous peritonitis. (1) He is not of the age group of this disease unless it was an acute flare up. (2) He does not give the physical findings of a tuberculous peritonitis. (They usually have abdominal pain—this is not described here.) They are usually adolescent or young adults. All of this discussion previously, represents this patient's primary disease and, I feel undoubtedly is responsible for his death.

SUMMARY

A case of (1) *a chronic constrictive pericarditis, probably secondary to tuberculosis;* (2) *a tuberculous pneumonia;* (3) *and/or miliary tuberculosis;* (4) *possibly a tuberculous peritonitis.*

There are certainly other diseases worth commenting on. I have not discussed the possibility of his having had amyloidosis. There may have been amyloid changes in his kidneys, liver, and spleen secondary to a prolonged acid-fast infection. I would dismiss the idea of his having had amyloidosis as a primary disease by the fact that he has a Congo red test that shows no dye absorbed in one hour. One would expect a large amount of Congo red to be absorbed in primary amyloidosis.

Where there is a history of weight loss, enlarged heart, distended abdomen and enlarged spleen and liver, I think it would be wise to consider carcinoma. If he had a carcinoma I do not know where it is. There are no findings that point specifically to any one area as the seat of his trouble, unless it is pulmonary, and in that case bronchogenic carcinoma should be eliminated. He has had x-rays but there is nothing to indicate on the protocol that anything was found other than a pleural effusion.

Pick's disease is a consideration. There are some advocates who feel that it can be a clinical entity. Pick's disease is a serositis in which there are effusions of the pericardium, peritoneum, and pleura. I do not know what the other part of the picture is, whether they are afebrile or not, for instance. Chances are that they are. It does end terminally in enlargement of the liver and spleen. I think on the basis of that fact that it is a relatively rare condition, if it actually exists at all as an entity, and we should not consider it too seriously.

Boeck's sarcoid should also be mentioned as a consideration. It is characterized by skin and bone lesions that are not mentioned here. However, pulmonary pathology and lymphadenopathy are also encountered both in this disease and in this case. A skin biopsy was not done. This could have aided in establishing this diagnosis.

There are some other findings that are of incidental interest. He has renal findings that I have not even discussed, it was shown that he had a positive urine culture of atypical coliform and E. coli. This could be secondary to his debilitation and prolonged bed rest that he was on in the hospital. I feel that the albuminuria could be explained by the presence of debilitation and some renal damage from the miliary process.

The A/G ratio alteration and the abnormal prothrombin percentage and cephalin flocculation tests are certain evidence of hepatic insufficiency, all of which are conceivably consistent with miliary tubercles both primarily and secondarily since their presence would impinge on normal liver cells and alter the function of the organ as a whole.

PATHOLOGICAL DIAGNOSIS

Chronic Constrictive Pericarditis
Miliary Tuberculosis

"POTENTIAL" INGUINAL HERNIA*

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INTRODUCTION

This paper is a report on 166 workers with normal inguinal rings and on 206 workers with enlarged inguinal rings, in which—of the group with normal inguinal rings—9 developed hernia, and of the group with "potential" hernial rings only 4 developed hernia.

The inguinal hernia is one of the oldest lesions known to man, having been portrayed in the ancient art of Egypt and Greece.[1] In 900 B. C. the Phoenicians treated hernia by girdle and compression.[2] Hippocrates recom-

*Read before the Medical Society of Delaware, Rehoboth, September 15, 1948.
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mended the use of plaster as treatment for hernia, and Celsus in the First Century A. D. practiced ligature and excision of the unopened sac.[2]

The word "hernia" was derived from the Greek term meaning a branch or outgrowth—which was the ancients' conception of hernia.[1] In the Middle Ages this belief was displayed by one which considered hernia as a rupture, or "bruch," of some of the retaining structure, implying that all inguinal hernias were acquired.[1] Biologically, the hernia is the result of the assumption of the erect posture, as quadrupeds do not have hernia.

The abdominal, or internal, inguinal ring is oval in shape, and it is located in the transversalis fascia. There is no means on physical examination of determining the size of this ring. With the persistence of the patent funicular process in the male, there is provided a potential hernial sac for an escape of the viscera downward through the subcutaneous or external inguinal ring. This ring is an interval in the aponeurosis of the external oblique muscle. The aperture is triangular in form and usually measures from base to apex 2.5 cm. and transversely 1.25 cm. The importance of the measurement of this external ring is controversial. Jacob N. DaCosta, Professor of Medicine at Jefferson Medical College in the 1870's, stated, "In a healthy male the external ring should admit the tip of the little finger but not the end of the index finger. If the end of the index finger can be entered into the ring, that aperture is dilated, and even if there is no hernia in the canal, in the future a hernia will probably descend."[3] Lawrence S. Fallis, a Professor of Surgery at Henry Ford Hospital in Detroit, has stated, "If the external inguinal ring is smaller than the tip of the index finger, there is usually no hernia."[4] The end of the average little finger in the male adult measures approximately 1 cm., while the index finger is approximately 1.5 cm. In this report a "potential" inguinal hernial ring is one that has a transverse diameter of 1.5 cm. or larger, with or without impulse. Probably many physicians will say that 1.5 cm. is a normal ring size. The physicians who examined the workers reported here felt at the time of the examinations that an external ring with a measurement of 1.5 cm. was enlarged.

It is usually an easy procedure to estimate the external ring, but various examiners' interpretation may vary by approximately ½ cm. In the cases reported here, most of the reexaminations have been done by the same physician who made the initial estimation of the size of the external ring.

The statistics in this report are not infallible, but it is the hope of the writer to stimulate more interest in this pertinent problem. Many an individual has been rejected from an industrial position because of the report by a physician of a "potential" inguinal hernia.

In 1941 a request was made to a number of industrial plants for a report on the size of inguinal rings in workers. From this report we have rechecked all employes who have remained engaged in industrial work for from five to fifteen years. In this paper the individuals with normal inguinal rings and those with "potential" hernial, or enlarged, rings are included. There are more cases reported in the group with "potential" rings, as some plants reported only on that group. Of the individuals with normal rings less than 1.5 cm. in diameter, there were 166. Nine of these men, or 5.4 per cent, developed hernia. The average age at which the hernia developed was 54. The following chart shows the breakdown into age groups with incidence of hernia.

CHART No. I

NORMAL INGUINAL RINGS

Age Group*	Number of Employes	Number of Age Group Developing Hernia	Per Cent of Age Group Developing Hernia
21-30	12	0	0.0
31-40	58	0	0.0
41-50	42	4	9.5
51-60	36	3	8.3
61-70	17	2	11.7
71-80	1	0	0.0
All Ages	166	9	5.4

*Age is that at last examination or year hernia developed

Of these 9 men who developed hernia, all with the exception of 2 had external rings recorded as 1 cm., and those two were 1.2 cm.

In the group with enlarged rings (1.5 cm. or larger, with or without impulse) there were 206. Four of these workers developed hernia. The average age at which hernia de-

veloped was 36 years. The following chart shows the breakdown into age groups with incidence of hernia.

CHART No. II

"POTENTIAL" HERNIAL RINGS

Age Group*	Number of Employes	Number of Age Group Developing Hernia	Per Cent of Age Group Developing Hernia
21-30	9	0	0.0
31-40	64	3	4.5
41-50	63	1	1.5
51-60	45	0	0.0
61-70	21	0	0.0
71-80	4	0	0.0
All Ages	206	4	1.9

*Age is that at last examination or year hernia developed

The 4 men who developed hernia had rings of 1.5 cm. in diameter, and only one had an impulse in the inguinal ring.

M. B. Landers, medical director of a large industrial organization in Detroit, Michigan, reported on 3,680 men in industrial work. There were 365, or 9.9 per cent, who showed inguinal hernia.[5] D. B. Moss, Chief Surgeon of the Burlington Railroad, reported that of 700 employes free from hernia on employment, 15 or 16 per cent had developed hernia when checked after a 10 to 15 year period.[6] A report was made in 1943 by Lieut. Comdr. Francis P. Gardner on 24,934 preemployment examinations in 1926. In this group of civilian employes 53, or .22 per cent, developed hernia. A further breakdown shows 21,521 had normal rings with 42, or .176 per cent, developing hernia, and 3,413 had relaxed inguinal rings with 11, or .322 per cent, developing hernia.[3] There was no definition as to the size of the "normal" or the "relaxed" inguinal ring.

The hernia may be classified into one of three types: 1) the true congenital which develops early in life because of the failure of normal obliteration of the peritoneal canal; 2) the hernia which may be congenital and develops in man after 40 years of age; and 3) the true traumatic hernia, which is least common and has a history of a tearing of the inguinal ring associated with edema and ecchymosis.

The hernia is most often found in men over forty years of age and is more common in industrial employes and farmers. In this laboring class one would anticipate a strengthening of muscles, fascia, and tendons. Concurrently with this tension of the abdominal muscles there are fluctuating increases in intra-abdominal pressure and progressive degenerative processes that normally occur in the muscles and fascia after the third and fourth decades. Thus there may develop over a period of years a gradual out-pouching or stretching of the peritoneum through the abdominal inguinal ring. In most cases the hernia is discovered by the physician on a physical check-up, or it is found by the individual while bathing.

According to recent reports 5 per cent of the total male population have hernia,[3] or 10 per cent of the male population between the ages of 20 and 70.[4] It is possible on a routine or even a careful examination to miss a hernia. There are cases with small external rings in which coughing alone will not force down the sac. Most of these hernias are the early stages in the development of the true inguinal rupture. The peritoneal protrusion is in the inguinal canal, and only with marked or constant increase in intra-abdominal pressure will the hernia be forced through the external ring. In time this repeated procedure will dilate that ring.

CONCLUSIONS

The term, "potential inguinal ring," is frequently found in literature, but there is no definition of what diameter determines this classification. From the findings in this report, the belief is held that such a term has little significance. It may be more important to determine the strength of the abdominal muscles, the body build, the degree of adiposity, or to measure the inguinal ligaments.[7]

It has been noted frequently that hernia tends to be bilateral. In the literature it is reported that with a hernia developing unilaterally between the ages of 20 and 30 years, there is a 12.6 chance of having one develop on the other side within 10 years.[5] This finding upholds the theory of congenital weakness and decrease in muscle and fascial strength.

In the group reported with the larger external rings, approximately 37 per cent had subcutaneous rings measuring 2 cm. or larger. None in this group developed hernia. This finding bears out the statement, "A large diameter of the fascial ring does not favor

the existence of a hernia any more than a large mouth will permit more food to pass through into the esophagus."[1]

SUMMARY

(1) Report on 166 workers with normal inguinal rings and on 206 workers with enlarged inguinal rings.

(2) Of the group with normal inguinal rings 9 developed hernia, and of the group with "potential" hernial rings only 4 developed hernia.

(3) It is herein concluded that there is no justification for the employment of the term, "potential inguinal hernia."

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Pit-Thyrocas (Formerly Anti-Obese) and Glan-Obese (Gerritt Company)

The Gerritt Company of Los Angeles, California, circularizes physicians with a form letter containing excerpts from "unsolicited" testimonials of other physicians to promote the sale of a tableted "shotgun" mixture of drugs for obesity under the name Pit-Thyrocas. This advertising makes it plain that the name of the product was formerly Anti-Obese and that the firm claims it to be "An Ethical Product, Sold Only in an Ethical Way." The firm proposes to send to the recipient as a special 30-day offer, five prescription labeled boxes of 100 tablets each at a net cost of $3, for which the selling price of $17.50 ($3.50 per box) is quoted—leaving a profit of $14.50! This remarkable offer to physicians also includes a free "diet schedule" sheet, in each box if desired, cleverly designed to oversimplify the management of patients who desire to reduce weight. The product is backed by the familiar guarantee, "Your Money Back If, After Trial, You Are Not Satisfied."

The firm's advertising leaves little to the judgment or, indeed, intelligence of the physician. It includes the following formula for Pit-Thyrocas: "Thyroid Des. 5/8 gr.; Pituitary Whole Des., 1/10 gr.; Powd. Ext. Phytolacca Berries, 1/4 gr.; Phenolphthalein, 1/4 gr.; Precip. Cal. Carbonate, 3 gr.; Extract Cascara Sagrada, 1/4 gr." This is identical with the formula previously promoted under the name Anti-Obese and is subject to the same alteration: "Occasionally these tablets are too laxative, so we make them with and without Phenolphthalein—same price, color and size." This makes the formula identical with "Thyrop" of the Cole Chemical Company, which has been the subject of adverse comments in a previous Council Report.[1]

Although also sold to pharmacies for filling prescriptions, an appeal to dispensing physicians is made as follows: "This is one tablet which many physicians prefer to dispense in order to observe the effect on the heart," —and then, there is a profit motive: $2.90 per hundred at the special rate; $1.75 to $2.45 per hundred at regular list prices, "or any other price you may choose to charge!" The form letter further has this to say concerning the mixture:

"Pit-Thyrocas Tablets reduced weight because of the well-known action of Thyroid and Pituitary in hyperthyroidism. Poke Root lessens the desire for food. Calcium Carbonate partially alkalinizes the HC1 in the stomach. Phenolphthalein and Cascara Ext. make it the perfect eliminant for treating adiposity."

The foregoing quoted explanation of the mixture is an attempt to lend a "scientific" aspect to the product. Well trained physicians should not be "taken in" by this verdant verbiage! For the sake of the less wary, it may be stated that neither thyroid (which is active orally) nor whole pituitary (which is inert by mouth) is presently considered useful in the treatment of obesity except when this is secondary to hypothyroidism, in which cases thyroid alone would be sufficient and oral pituitary wholly ineffective. In fact, thyroid should be considered potentially harmful and dangerous in persons with a normal basal metabolism. Phytolacca-Poke (puke) berries or root, formerly employed as an antirheumatic of dubious value, should be relegated to folklore medicine, where it belongs.

1. Drugs for Obesity, Report of the Council on Pharmacy and Chemistry, J.A.M.A. 134: 527-529 (June 7) 1947.

There are superior agents now available for depression of the appetite when this action is helpful in eliminating the habitual excessive intake of food that characterizes most cases of obesity. Just why it should be desirable to buffer the hydrochloric acid of the stomach in such cases, unless there is a definite hyperchlorhydria, is not explained by the firm. Possibly the idea is to interfere with protein digestion in the stomach and so enable the patient to eat more, but absorb less. In any case, 3 grains of calcium carbonate would do very little buffering. If it is intended to reduce appetite, its effectiveness for this purpose is nil. Hurrying the passage of partially digested food through the intestinal tract by means of cathartics at most interferes a little with caloric intake at the level of the intestine instead of at the table. The increased elimination of water by the use of cathartics, if it is sufficiently severe, produces some temporary reduction of weight, but not of "fat." The formula thus represents an irrational approach to the treatment of obesity in which the importance of correction of habits of overeating is mostly ignored. The "diet schedule" offered also is unscientific in that it is based on a qualitative rather than a quantitative restriction of caloric intake.

The form letter does not stop with Pit-Thyrocas. The closing paragraphs promote what is described as a newer product designated "Glan-Obese Pluraglandular Solution." Concerning this mixture, it is stated "that an additional glandular product was necessary to satisfactorily treat such cases of obesity where Pit-Thyrocas Tablets alone would not produce the desired effect." This admission may come as somewhat of a shock to the gullible reader of the testimonials for the tablets! An accompanying leaflet titled, "Glan-Obese No. 1," states:

"Each 2 cc. of this formula contains the aqueous extractives of the highest quality Glandular substances as follows:

Ovarian Substance	40 gr.
Anterior Pituitary Substance..	30 gr.
Suprarenal Cortex Substance..	30 gr.
Thyroid Substance	3 gr.
Posterior Pituitary	3 I. U.
Chlorobutanol	0.5%"

Net wholesale prices are also quoted for 30 and 60 cc. vials of this mixture. It is blatantly recommended by the firm for the parenteral treatment of obesity (to be administered alternately or in conjunction with the oral Pit-Thyrocas) and for "Multiple Glandular Deficiency Troubles in the Male and Female." The form letter concludes with the additional information that physicians "are using Glan-Obese solution for treating such painful uterine conditions as Amenorrhea and Dismenorrhea." Yet with the utmost candor, it is stated in the Glan-Obese No. 1 leaflet that "Hormone activity in this solution has not been investigated." This statement stands in curious contradiction to other statements in the leaflet that, "Most authorities agree that lack of gland functions is the direct cause of obesity in most cases" and that "Glan-Obese is a 100% gland stimulant." The claim that lack of gland function is the primary cause of most cases of obesity is absolutely false and not supported by any scientific authority.

The advertising for Glan-Obese goes on with the maze of contradictions in the statement, "It has become a well established fact that parenteral injections of glandular extracts are far more potent in reducing weight, than the same products are when taken orally." This of course means nothing when applied to the gland "substances" in the mixture which are not potent hormones. The physician might well ask why the firm offers the oral preparation of Pit-Thyrocas Tablets if the last quoted statement has any significance. He may well ask why Glan-Obese solution, also glibly recommended for the treatment of multiple glandular deficiency, is of any value for this purpose or in obesity due to "lack of gland functions," when the product is said not to have been tested for hormone activity! Most physicians will realize that thyroid is active by mouth and is almost never injected even in the form of thyroxine for the treatment of severe hypothyroidism. It will also be realized that when one wishes to inject hormones, potent extracts or synthetic compounds, assayed for hormone activity, must be employed to obtain therapeutic results. A "shotgun" mixture of untested "substances" has no value given either by mouth or by injection.

It seems obvious that the Gerritt Company promotes the sale of Pit-Thyrocas and Glan-Obese for obesity wholly on a commercial appeal to profit. This is not new in the annals of questionable advertising for worthless remedies. In this particular instance it seems incredible that the manufacturer could induce well trained and thoughtful physicians to prescribe these products, but the protests which have been received at the Council office make it advisable to issue this statement so as to dispel any doubt concerning the character of these two "remedies" for obesity. *J.A.M.A.*, July 2, 1949.

+ Editorials +

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VOL. 21 JULY, 1949 No. 7

POTATOES IN POLITICS!

The politicians with potatoes in their paunches and in their portfolios didn't know what punches they were pulling when they put potatoes in politics.

Pulling $2,000,000 annually out of the people's pockets to pay potato planters parity-plus pyramids the popular price, impoverishes the dinner pot and pauperizes the general population in favor of the potato plutocrats.

It requires 1,000,000 common people paying an income tax of $200 to pacify the 28,444 potato planters. The downtrodden planters in Rhode Island unloaded enough potatoes on the taxpayers to average $23,206 per grower. In Massachusetts $12,229, in Maine $9,825, in New York $13,169 and so on down the line.

The wisdom of our government is beyond finding out.

Apparently having demonstrated their expertness in the handling of potatoes the bureaucrats are ready to take on the physicians. Compared to the cost of socialized medicine, everything else can be counted as small potatoes.

Editorial, *J. Okla. S. M. A.*, July, 1949.

PROGRESS IN MEDICAL CARE—AND NO LAW WAS PASSED

Had anybody a few years ago prophesied that a city the size of Baltimore could shortly do away with its hospital for communicable diseases, he would have been thought mad. Yet such has come to pass with Mayor D'Alesandro's announcement that Sydenham will be closed.

This revolutionary change is not due to socialized medicine or any other experiment in medical-care administration. It is due solely and entirely to the discovery of the remarkable remedial effects of the antibiotic drugs, including the sulfa group, penicillin, aureomycin, and the like. Through their use patients at Sydenham are enabled to cut short their stay. Others who would have been patients can be treated with the drugs in their homes.

It was only very recently that officials of Sydenham and of the Health Department, after careful study, were convinced that the limited work that Sydenham now does could be taken care of by the general hospitals. Baltimore is fortunate in having a capable advisory board to give the Health Department counsel in the making of such momentous decisions. Therefore when Mayor D'Alesandro announces that Sydenham will be abandoned, but that the public medical services of the city will not suffer, his statement is backed up by the sound judgment of the medical profession.

In addition to caring for patients with communicable diseases the staff at Sydenham also is engaged in important research. It is reassuring to learn that Sydenham will be closed only after provision has been made for taking care elsewhere of this work too.

Here we have a striking example of progress in medical care which not only improves the quality of treatment but does so with tre-

mendous reduction of cost. This change is coming about not through the passage of a law, but through the result of normal medical progress. It is another potent warning that in trying to raise the national standard of medical care by Act of Congress we are on the wrong track.

Editorial, *Baltimore Sun*, June 23, 1949.

DR. BEATTY HONORED

Dr. Gerald A. Beatty of Wilmington was re-elected as Governor of the American College of Chest Physicians for the State of Delaware at the Fifteenth Annual Meeting held in Atlantic City, New Jersey, June 2-5, 1949.

THE JOURNAL congratulates our eminent colleague on not only this re-election, but also that of President of the newly constituted Board of Health for Wilmington.

MISCELLANEOUS

A. M. A. Atlantic City Convention

The total registration of over 13,000 physicians exceeded any previous convention of the A. M. A. with the exception of the Centennial Session held in Atlantic City two years ago. To this figure should be added 14,000 guests (wives, residents, interns, medical students, nurses and technicians) and 3700 representatives of technical exhibits.

The weather throughout the five-day meeting was ideal, thereby adding to the pleasure of the registrants.

GRASS ROOTS CONFERENCE

The fifth National Conference of County Medical Society Officers was held on Sunday, June 5th, with morning and evening sessions. Among the important subjects discussed at the conference were Emergency Care, Indigent Medical Care Plans, and the National Education Campaign of the A. M. A. The evening session was highlighted by the talk of John L. McClellan, U. S. Senator from Arkansas, and Mr. Clem Whitaker of the firm of Whitaker and Baxter, Director of the A.M.A. National Education Campaign.

STATE SOCIETY OFFICERS MEET

On Sunday afternoon the Conference of Presidents and other officers of the State Medical Associations took place at which time the British National Health Service, the Relations of the State Society to the A.M.A., and the State Compulsory Disability Compensation Programs were discussed. Two outstanding speakers spoke on Socialized Medicine in England—Dr. Ralph J. Gampell, a former English doctor now interning in the U. S., and Mr. Cecil Palmer of London now on a lecture tour of this country. Both of these speakers left unmistakable evidence of the effect of the present medical system in England on the quality of medical care and the general economy of the nation.

HOUSE OF DELEGATES MEETINGS

Sessions of the House took place on Monday, Tuesday and Thursday with F. F. Borzell of Philadelphia ably presiding as Speaker. Among the important matters which occupied the attention of the House were compulsory health insurance, medical care of the veteran, voluntary medical care plans, the practice of medicine by hospitals, and the National Education Campaign of the A.M.A., directed by Whitaker and Baxter. Various reports and resolutions dealing with these and other matters were presented and referred to specific Reference Committees for hearings and recommendations. Among the most important Reference Committee was that dealing with Legislation and Public Relations headed by William Bates of Philadelphia. This Committee presented a very excellent report and the House took favorable action on the following recommendations emanating therefrom:

1. Proper safeguards should be set up with regards to the registration of birth certificates.
2. Expressed opposition to pending Federal legislation providing for diagnosis and treatment of physical defects among school children irrespective of the economic level of the child. This vicious legislation has already been passed by the Senate and is now being considered by the House.
3. Appointment of a special committee by the Board to arrange a conference among various Congressmen to develop legislation which would meet the objectives of the A.M.A. 12-point medical care program.
4. Questionnaire to be sent to all Fellows and members of the A.M.A. seeking their

reaction on a confidential basis to the A.M.A. Health Program.

5. Cooperation of the A.M.A. in the Diabetes Detection program of the American Diabetes Association.
6. Approval of the Code of Ethics of the World Medical Association.
7. The name of the A.M.A. should be identified as the sponsor of the National Education Campaign being undertaken by Whitaker and Baxter.
8. Opposition by the A.M.A. to the activities of the Federal Government in the field of private enterprise.
9. Opposition by the A.M.A. to the inclusion of physicians and other self-employed persons under the present Social Security Act.
10. Federal Employees under Federal Compensation Acts should be entitled to free choice of physician.
11. Appointment of a special committee on Veterans Affairs to study the medical problems of the veteran especially with reference to non-service connected benefits.

ENDORSEMENT OF LAY SPONSORED MEDICAL CARE PLANS

The House took initial steps to recognize medical care plans operated by lay groups in a special report prepared by the Council on Medical Service embodying a set of 20 principles to be used as a guide by both State and County Medical Societies in evaluating these plans seeking A.M.A. endorsement. This action clearly recognized the important part which lay insurance groups should play in developing plans covering medical, surgical and hospital insurance benefits. Up to the present time, the A.M.A. has only endorsed medical sponsored plans.

WHITAKER & BAXTER

Mr. Clem Whitaker and Miss Leone Baxter appeared before the House of Delegates and rendered comprehensive reports concerning the organization and activities of the National Education Campaign since the first of the year. It was quite evident that their efforts were beginning to show positive results as evidenced by the fact that over 800 national lay organizations had already come out officially in opposition to compulsory health insurance. In addition, a great deal of effective educational campaign material had been distributed to physicians and the lay public, as well as members of Congress, in answer to the government program and emphasizing the fact that voluntary plans can do the job better. Tribute was paid to the cooperation of individual physicians in carrying out the objectives of the campaign but the interest of many additional physicians was solicited. Attention was called to the fact that the recent announcement from Washington that no action would be taken on the Government's health program should not mislead us in believing that the battle is won and that we can relax our efforts. This will afford the profession much-needed additional time in which to further the educational campaign in anticipation of the question of compulsory health insurance again coming up in the next Congress.

DR. FISHBEIN'S ACTIVITIES CURTAILED

The Board of Trustees in a special supplemental report announced that the future talks, writings, and interviews of Dr. Fishbein will be limited to scientific matters, and that plans were also being formulated for the training of a new editor and the retirement of the present Editor. However, this action in no way casts any reflection on the valuable job performed by Dr. Fishbein for the A.M.A. during his 37 years' affiliation with the Association.

CONFERENCE ON HEALTH CALLED

Announcement was made that the A.M.A. will sponsor a national Health Conference in the late summer or fall to implement the Association's 12-point health program. This was indicative of the desire of the A.M.A. to adopt a more positive approach in its present aggressive fight against Compulsory Health Insurance.

The Delegates approved the creation of a new Section on Physical Medicine and Rehabilitation as well as suggesting to the Board that it consider the formation of a single membership in the A.M.A. to take the place of the present arrangement whereby members of constituent County Societies must apply for Fellowship in the A.M.A. to receive full privileges of membership in the A.M.A.

One fact stood out in the deliberations of the House of Delegates—namely, the unanim-

ity of opinion that the A.M.A. should wage a continuous and aggressive fight against Compulsory Health Insurance or Socialized Medicine, and that full support must be given to the extention of health insurance on a voluntary basis. One can only appreciate the democratic procedure of the House by watching it in session.

DR. HENDERSON BECOMES PRESIDENT-ELECT

Dr. Elmer Henderson of Louisville, Ky., a member of the Board of Trustees of the A.M.A. for the past 10 years, was elected unanimously as President Elect of the Association to take office next June in San Francisco. Dr. Ernest E. Irons was installed as President for the period 1949-50 at a special meeting held on Tuesday evening, June 7th, in Convention Hall at which time the Distinguished Medal was awarded to Dr. Seale Harris, Professor Emeritus of Medicine at the University of Alabama.

Other officers elected were Dr. James F. Norton of Jersey City, Vice-President; George F. Lull, Secretary; Josiah J. Moore of Chicago, Treasurer; F. F. Borzell of Philadelphia, Speaker; James R. Reuling of Bayside, New York, Vice-Speaker. Dr. Louis H. Bauer of Hempstead, L. I., was re-elected a trustee for five years and Dr. F. J. L. Blasingame of Wharton, Texas, was elected a trustee for a five-year period to succeed Dr. Henderson.

FUTURE MEETINGS

Chicago was selected as the site of the 1952 meeting, San Francisco having been already selected for 1950 and Atlantic City for 1951. The 1949 interim session will be held in Washington, D. C., and the 1950 interim session in Denver, Colorado. It is hoped to hold the 1951 interim session in Houston, Texas.

RELIGIOUS SERVICES

Continuing the precedent set at the Centennial Session two years ago a special all-faiths religious service was held in Convention Hall on Sunday, June 5th, participated in by Francis Cardinal Spellman of New York, Rabbi Morris S. Lazaron of Pikesville, Md., rabbi emeritus of the Baltimore Hebrew Congregation, and Rev. John McCartney, Director of Chicago Sunday Evening Club.

EXHIBITS WELL ATTENDED

The entire first floor of Convention Hall was devoted to technical and scientific exhibits. This year represented the fiftieth anniversary of the scientific exhibit which was founded in 1899 by Dr. Frank B. Wynn. Proper recognition of this fact was paid during the course of the Convention. The overflowing number of persons constantly on the exhibit floor was ample evidence of the attractiveness of the exhibits.

Reports indicate that all of the general scientific and section meetings had capacity audiences.

Philadelphia Med., June 18, 1949.

Thought For Today

In a twelve-month period in 1945-46, the United States sent delegations to over 100 *official* international conferences and meetings. Our spokesmen were heard in Montevideo and Copenhagen, in Moscow, in Bermuda, in Seattle. These are some of the subjects (many subheads and all nongovernmental conferences being excluded) on which the United States expressed its policy internationally in that one year:

Aerial Law
Agriculture
Astronomy
Aviation Routes
Cereals
Coal Mining
Copyright
Cotton
Development Works
Education
Films
Geodesy
German Assets
Health
Indians
Infestation
Inland Transportation
International Bank
Iron and Steel
Labor
Marine Navigation
Maritime Authority
Marine Labor
Metal Trades
Meteorology
Monetary Fund
Ophthalmology
Palestine
Penitentiaries
Petroleum
Protection of Childhood
Public Health
Radio Frequencies
Refugees
Relief
Reparations
Repatriations
Rubber
Sanitary Engineering
Scientific Unions
Seeds
Social Security
Social Service
Statistics
Sugar
Tangier
Telecommunications
Whaling
Wheat

—From "American Education and International Tensions," a report of the Educational Policies Commission of the National Educational Association.

Hypnotism For Entertainment Is Dangerous Performance

Hypnotism is not an innocuous performance to be used for entertainment, warns a medical consultant of the *Journal of the American Medical Association.*

"Hypnosis should not be allowed outside of the medical profession, and laws are needed, forbidding the use of hypnosis for entertainment purposes," he advises in the current (June 25) issue.

"A public performance has the probability of doing great damage. Neurotic symptoms can be created readily by direct suggestion in the average adult. But since children are more suggestible than adults, the potential harm is even greater.

"In competent hands hypnosis has no harmful effects, but where it is utilized for nonsensical and dramatic effects, and where removal of symptoms is attempted without some understanding of the dynamics of the subject's personality, hypnotized persons may be adversely influenced.

"Since many youngsters have a sense of insecurity and are therefore potentially neurotic, they have more serious problems in interpersonal relationships. When they are exposed to an injudiciously applied hypnotic trance, they may become acutely upset."

Dyes Used to Mark Diapers Are Dangerous to Babies

Use of aniline dyes, those derived from coal tar, to mark diapers may cause serious poisoning in babies, points out an editorial in the current (June 25) *Journal of the American Medical Association.*

Seventy-two cases of poisoning in babies from the dyes have been reported, including five deaths, the editorial says, adding:

"Prevention of such accidents is simple. If the diapers are boiled after they are stamped and thoroughly dried before use, the dye becomes fixed and absorption does not occur. The ideal methods of prevention would be the use of nontoxic dyes, but, unfortunately, vegetable pigments, charcoal, and silver nitrate lack the permanence required for marking clothing in large institutions."

OBITUARY

ALLAN V. GILLILAND, M. D.

Dr. Allan V. Gilliland, 47, practicing physician at Smyrna for the last six years and first superintendent of the Welfare Home there, died suddenly of a heart attack at his home on June 29, 1949.

Appointed by the State Welfare Commission as superintendent and director of administration when the Welfare Home was opened in 1933, Dr. Gilliland served in that capacity for ten years, when he resigned to return to private practice. Before coming to Delaware he was connected with the Rockview Penitentiary at Bellefonte, Pa. He had practiced medicine in Michigan and in this state before going to the Welfare Home.

Dr. Gilliland was born at Stratford, Ontario, Canada, in 1901, and received the degree of M. D. from the University of Western Ontario in 1926. He was licensed in Delaware in 1932. He became a naturalized citizen while at the Welfare Home.

Dr. Gilliland was secretary of the Medical Society of Delaware in 1938. At the time of his death he was still a member of the Society, and of the New Castle County Medical Society, and of the American Medical Association.

Surviving Dr. Gilliland are his wife, Mrs. Lois Garrett Gilliland, daughter of the late Mr. and Mrs. Philip L. Garrett of Hockessin; a daughter, Miss Ruth Louise Gilliland of Sudbury, Canada; his father, George R. Frank Gilliland of Canada; a sister, Lulu M. Gilliland of Detroit, Mich. His mother was the late Martha Graham Gilliland.

The funeral was held at Smyrna on July 2, 1949, the Rev. Walter Hards, pastor of the Presbyterian Church of Smyrna, officiating. Interment was in Friends Burying Grounds at Hockessin, Delaware.

1789—MEDICAL SOCIETY OF DELAWARE—1949

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R. O. Y. WARREN, *President-elect*
A. D. KING, *Vice-President*
D. D. BURCH, *Secretary*
CHARLES LEVY, *Treasurer*

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Alternates (1949): E. M. Bohan, I. M. Flinn, Jr., A. D. King, C. E. Maroney, E. T. O'Donnell, W. M. Pierson, D. J. Preston, W. T. Reardon, J. A. Shapiro, O. N. Stern, J. W. Urie.

Delegates (1950): C. W. Bancroft, N. L. Cannon, I. L. Chipman, A. M. Gehret, A. L. Heck, J. W. Hooker, C. T. Lawrence, Charles Levy, C. L. Munson, M. B. Pennington, J. C. Pierson, S. H. Stradley.

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C. J. PRICKETT, *Secretary-Treasurer*, Smyrna.

Delegates: Henry V'P Wilson, Dover, I. J. MacCollum, Wyoming.

Alternates: J. S. McDaniel, Dover, Hewitt W. Smith, Harrington.

Censor: R. W. Comegys, Clayton.

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SUSSEX COUNTY MEDICAL SOCIETY

Meets Second Thursday

JOHN W. LYNCH, *President*, Seaford.
JAMES E. MARVIL, *Vice-President*, Laurel.
LESLIE M. DOBSON, *Secretary-Treasurer*, Milford.

Delegates: Bruce Barnes, Seaford; T. J. Tobin, Milton; W. G. Hume, Selbyville; O. A. James, Milford.

Alternates: Wilbur Ellis, Laurel; R. L. Klingel, Rehoboth; A. C. Smoot, Georgetown; L. L. Fitchett, Milford.

DELAWARE STATE DENTAL SOCIETY

R. R. WIER, *President*, Wilmington.
CLYDE COX, *First Vice-President*, Newark.
JOSEPH MACK, *Second Vice-President*, Seaford.
NORBERT GLADNICK, *Secretary*, Wilmington.
H. H. MCALLISTER, *Treasurer*, Wilmington.
P. E. MUSSELMAN, *Delegate A.D.A.*, Newark.
CLYDE NELSON, *Alternate A.D.A.*, Milford.

DELAWARE STATE BOARD OF HEALTH

J. D. Niles, M. D., *President*, Middletown; Mrs. F. G. Tallman, *Vice-Pres.*, Wilmington; W. B. Atkins, D. D. S., *Secretary*, Millsboro; Bruce Barnes, M. D., Seaford; Mrs. C. M. Dillon, Wilmington; J. B. Baker, M. D., Milford; Mrs. Alden Keane, Middletown; E. R. Mayerberg, M. D., Wilmington. Edwin Cameron, M. D. *Executive Secretary*, Dover.